LETTERS TO THE EDITOR

CORONARY SPASM AND ORGANIC STENOSIS

MacAlpin found that in five of six cases of spontaneous or drug-induced dynamic coronary occlusion that caused attacks of variant angina, the obstruction occurred at the site of a well defined organic stenosis,[1] and also that in 90 percent of cases spasm occurred at the site of an organic lesion.[2] The existence of spontaneous coronary spasm or constriction is no longer doubted by anyone. During the last 10 years or so I have treated patients who manifested a spasm of accommodation with various concomitant symptoms of imbalance in the autonomic nervous system. Even young people who are otherwise completely healthy often have very severe pains in the region of the heart. I have found that when the spasm is released, such pains disappear. This observation has obliged me to ponder the possibility that severe constriction of long duration could disturb nutrition of the blood vessels and that the resulting tissue damage could produce the preconditions for sclerotic change.

I am prompted to ask whether the organic lesion itself may not be the consequence of a primary spasm at the same site. In other words, may not the spasm be the etiologic cause of (coronary) sclerosis? The next stage will be to ask "why are some people's coronary arteries so apt to vasoconstrict?" [3]

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REPLY

Having never previously heard of spasm of accommodation, I looked it up and found it described as attacks of convergence, accommodation and miosis of the eyes resulting in faulty vision. It is apparently an uncommon functional disturbance usually occurring in psychoneurotic persons.[1] That Viikari has encountered severe chest pains in some of his patients in association with spasm of accommodation is interesting. Electrocardiograms and esophageal motility studies on these patients during attacks of chest pain would be helpful in assessing the significance of the chest pain. I cannot find a reasonable explanation to connect spasm of the ciliary body and the coronary arteries except for an imbalance of autonomic nervous system function, which is apparently common in patients with spasm of accommodation but which I have not been able to detect in any of my patients with proved coronary spasm. To the best of my knowledge, none of the 70 patients with variant angina that I have seen have had a history of spasm of accommodation, although they were not asked about that specific entity.

The possibility that coronary spasm could injure the vessel wall and hence might itself be a cause of organic coronary artery disease was hinted at by Friedrich Kreysig in 1816,[1] and this hypothesis was more recently presented by Gertz et al. and by Marzilli and co-workers.[2,3] The merits of this hypothesis were discussed by Lown and DeSilva.[4] It is certainly not unreasonable to suppose that if coronary spasm occurs at the site of a preexisting pliable atheromatous lesion, the plaque could be disrupted and subintimal hemorrhage or endothelial rupture result—both of which could initiate acute coronary occlusion. It is known that severe spasm of cerebral arteries in human beings[5] and of coronary arteries in dogs[2] can result in damage to the vascular endothelium and smooth muscle. It is unknown whether spasm of a normal human coronary artery can also result in damage to the vessel wall, and whether this is *in reality* one cause of organic coronary artery disease and an explanation for the commonly observed relation of coronary spasm with sites of organic arterial stenosis.

Little is known of the normal determinants of vasomotion of large coronary arteries. Why some people's coronary arteries are so prone to vasoconstriction which can produce myocardial ischemia is still unknown.

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INABILITY OF A CHEST PAIN HISTORY QUESTIONNAIRE TO PREDICT CORONARY DISEASE IN THE EXERCISE LABORATORY

We have been using a Bayesian approach in our evaluation of the exercise test for the diagnosis of coronary artery disease. In an attempt to standardize our typing of chest pain, we

TABLE I

Questionnaire Scores for the Angiographic Groups

Angiographic Score	Questionnaire Score
0–3 (16)	8.4 ± 9.2
2–5 (7)	9.9 ± 4.4
>6 (23)	9.7 ± 10.9

Figures in parentheses indicate number of patients.

elected to use the questionnaire developed by the Health Insurance Plan of New York.[1] The type of chest pain, age and sex of the patient were then used to determine the pretest likelihood for coronary artery disease by the method of Diamond and Forrester.[2] The type of chest pain and the pretest likelihood for coronary disease were then compared with the angiographic findings.

There were 46 patients ranging in age from 32 to 72 years (mean 52.5). There were 33 men and 13 women. All patients had chest pain but no historical or electrocardiographic evidence for myocardial infarction. The questionnaire was filled out by a physician before the exercise test. Patients were then classed by their scores as follows: typical angina, >10 points and atypical angina, 0 to 10 points; negative scores were given for pain of noncardiac origin.

The arteriograms were reviewed without knowledge of the historical score. The coronary arteriograms were scored by the method of Friesinger et al.[3] Patients were placed in three groups: (1) a normal group with scores of 0 to 3 (with no lesion of 50 percent or greater), (2) a group with scores of 2 to 5 and at least one lesion of greater than 50 percent, and (3) a group of patients with scores of 6 or greater suggesting severe or multivessel disease, or both.

Correlations of the angiographic findings with the chest pain scores and pretest likelihood for coronary disease were performed using the multiple comparison analysis of Scheffe.

Table I gives the mean questionnaire scores for the angiographic groups. The scores do not correlate with the presence or severity of coronary disease. The pretest likelihoods for coronary disease also were not different for the angiographic groups (Table II). The percent of patients having coronary disease in the groups with chest pain was 62 percent (5 of 8) for the patients with pain of noncardiac origin, 67 percent (12 of 18) for those with atypical pain and 65 percent (13 of 20) for those with typical chest pain. For the men in this series, the percent of coronary disease in the groups with chest pain was

71 percent (5 of 7) for those with noncardiac pain, 69 percent (9 of 13) for those with atypical angina and 69 percent (9 of 13) for those with typical chest pain.

The classification of the chest pain by this questionnaire and the estimation of the pretest likelihood for coronary disease did not predict disease as assessed with angiography. In contrast, Chaitman et al.[4] reported that the pretest likelihood for coronary disease was 77 percent for patients with typical or probable angina but only 28 percent in patients having nonspecific chest pain. The clinical history of their patients was recorded by two physicians. Weiner et al.[5] also has reported that definite angina by history in men was associated with an 89 percent incidence rate coronary disease whereas those with noncardiac pain had a 22 percent incidence rate. For the success of the Bayesian approach the chest pain classification must be effective in predicting the pretest likelihood for coronary disease. The space given to the methodology of history-taking has been brief in the current publications on the Bayesian approach.[2,4–7] In our laboratory this has been a major problem in the successful clinical use of this approach.

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TABLE II

Pretest Likelihoods for Coronary Disease for the Angiographic Groups

Angiographic Score	Pretest Likelihood for Disease
0–3 (16)	48.6 ± 32.3
2–5 (7)	46.3 ± 19.4
>6 (23)	62.5 ± 27.9

Figures in parentheses indicate number of patients.

PULMONARY VALVE ECHOGRAM

The article by Starling et al.[1] validated our earlier report[2] that the presence of a B shoulder in the tricuspid valve echocardiogram is associated with elevation of right ventricular end-diastolic pressure. That observation is of particular importance in the assessment of total right heart function and in the interpretation of the pulmonary valve echo. The pulmonary valve echo will show loss of the A dip in the presence of pulmonary hypertension and sinus rhythm. However, the depth of the pulmonary valve A dip is not dependent on any absolute level of pulmonary arterial pressure, but probably reflects the relative right ventricular to pulmonary arterial end-diastolic gradient. A significant pulmonary valve A dip should not occur in the presence of significant pulmonary hypertension unless the right ventricular end-diastolic pres-